(the aura) and by a cry, and that he had tonic and clonic convulsions, foaming at the mouth, wounding of the tongue, insensibility to strong pricks, passing of the urine, and the characteristic aspect.

That the epilepsy depended upon the pathological condition of the stomach was shown by the cessation of the seizures from the time the stomach began to function regularly. The writer draws the attention to the absolute inefficacy of bromides to combat gastric epilepsy or to modify it; and likewise to the inefficacy of remedies for the catarrh and dilatation of the stomach, which yielded readily to repeated washings, which removed the accumulated secretions and stimulated to contractions the atonic and torpid muscular structure.

To sustain his diagnosis and the points that he makes, Dr. Zacchi cites a case of a woman whom he once saw with reflex nervous symptoms, face drawn and bathed with cold perspiration, eyes wide open and without expression, pulse slow and weak, unable to make an intelligent answer. She indicated with her hand that the stomach was the seat of her principal trouble. She recovered after she was made to vomit and had ejected a large amount of undigested food.

He also mentions a transitory reflex aphasia, the result of indigestion. G. P.

Society Reports.

AMERICAN NEUROLOGICAL ASSOCIATION.

Sixteenth Annual Meeting, held at Philadelphia, June 4th, 5th, and 6th, 1890.

(CONTINUED.)

Dr. PETERSON then read a paper, entitled: "A Case of Locomotor Ataxia associated with Nuclear Cranial Nerve Palsies and with Muscular Atrophies." (See page 450.)

Dr. C. K. MILLS thought that the case might be some peculiar irregular form of syringo-myelia.

Dr. E. D. FISHER suggested that the case was one of amyotrophic lateral sclerosis.

Dr. SACHS thought the case closely resembled one of his, in which the diagnosis was polio-myelitis, although there were more spmptoms of tabes dorsalis than in his case.

Dr. PETERSON did not think the case one of syringomyelia, owing to the very symmetrical distribution of the nuclear palsies, and because there was no anæsthesia of the upper extremities, face or trunk. There was no symptom of amyotrophic lateral sclerosis except the atrophy of certain muscles, which was subsequent to the disappearance of the knee-jerk. The speaker thought the case was probably one of extreme locomotor ataxia, with nuclear cranial nerve palsies and muscular atrophies dependent upon peripheral neuritis, as described by Déjerine.

A CASE OF COMPLETE PARAPLEGIA CURED BY OPERATION.

Dr. F. X. DERCUM presented a middle-aged man with the following history, in brief: The patient had, in 1887, suffered severe pain in the arms and shoulders. The pain had appeared to be referred to the principal nerve tracts. Soon after this he had lost power in his legs. Then about October of 1888, in addition to complete paraplegia, there was loss of sensibility, but with constricting pains about the upper portion of the chest. There was also complete paralysis of both sphincters. Examination had revealed the fact that the man's back was very painful over the third, fourth and fifth dorsal vertebræ. Thinking there might possibly be some local cause for the paralysis, the speaker had called others in consultation. It was agreed that the case was one in which trephining should be tried. The spines and arches of the first to the fifth dorsal vertebræ, inclusive, were accordingly removed. The dura was found somewhat abnormally resistant to the touch, and was opened. Adhesions existed between the dura and pia. After the operation, which the man had borne well, he had at once said that his pain was absent. A few days afterwards he was able to feel at the foot, when touched. Then he was sensible that his hands were cold, and was able in a few days more to move his toes. There had been a very gradual but steady progress towards complete recovery. He had also regained control of the sphincters. Whether the result was to be ascribed to local relief from pressure, or to reaction from the shock of the surgical operation, the speaker did not venture to suggest. The paraplegia was probably the result partly of pressure and partly of myelitis.

The President, in commenting on this unique case, characterized it as almost unprecedented.

Dr. PUTNAM said the case seemed to be one of unusual interest, and thought that they must all welcome such an advance in the surgery of this region. In a case of his, tumor of the spine was diagnosed, but operation was rejected. The rapidity of the development had suggested cancer of the vertebræ. He did not believe it was possible to tell from the severity of the symptoms, or the rapidity of their development, whether the conditions present were such as would contraindicate operation, and it ought to be done, even if the patient was in a pretty bad way, as a matter of investigation. He would call attention to the myelitis of Pott's disease and the paralysis due to œdema. In the case he had mentioned, the disease had turned out to be cancer. Horsely had reported among his cures one in which the disease had come on rapidly, and in which the local affection was carcinoma, but situated posteriorly. The case they had just seen would give a great deal of encouragement for the recommendation of such means of treatment.

Dr. V. P. GIBNEY said he was extremely delighted with the brilliant results in this case. From the way the patient now held his head he should be inclined to look upon the case as an old Pott's disease, and that the thickening found was the result of an inflammatory process within the bodies of the vertebræ. There might have been pressure myelitis from thickening around the vertebræ. The case was somewhat remarkable, considering the age of the patient. Surgeons had been chary of operating in these cases, though the operation was comparatively an easy one, at least simple in detail, and with the antiseptic and aseptic methods of to-day, it was practically impossible to have bad results. Recovery was what ought to be expected. Most of the cases so far operated upon, however, were of traumatic origin, old dislocations, fractures, and so forth. Of the cases of pressure myelitis in children a great many got well without treatment, while a certain number resisted all forms of intemperance. He had had a girl under treatment, by traction, for twelve months, and she had made a complete recovery. Still if it was practicable to cut down and do a laminectomy and get at the radical trouble, it would be much better.

The President said they would be glad to know to what extent the present position of the head corresponded with the position before rhe operation.

Dr. DERCUM said the position was then normal. He considered the present inclination of the head forward as due to loss of certain attachments of the trapezius muscles at their points of insertion into the spinous processes. It should be stated that there might have been a rheumatic element in this case affecting the meninges of the cord. The patient had described his pains as rheumatic, but they had subsided after the operation.

The President said he would like to ask Dr. Gibney as to the deformity in which there was extreme bending forward of the neck.

Dr. **GIBNEY** said these conditions sometimes obtained to an indefinite extent. It might be a senile hyphosis. Though it would perhaps be urged that Pott's disease at such an age was impossible, yet, as a matter of fact, there were quite a number in whom it developed after fifty or sixty, following a fall or injury. The position of this man's head might be due to loss of substance of the bodies of the vertebræ.

Dr. W. M. **BULLARD** thought the rapid advance in spinal surgery should lead to more definite conclusions as to the cases of Pott's disease suitable for operation. He was inclined to agree with Dr. Gibney that the case before them might be one of this nature. He did not think it necessary to avoid operation on account of the rapid development of symptoms. Extreme care should be observed in operations for Pott's disease in children. Many of these cases did well eventually if suitably treated by rest, and so on.

Dr. **SACHS** said he was gratified to hear the report of such a case as Dr. Dercum had presented. It went to show that many of the cases hitherto operated upon had been badly selected. It was difficult as a rule to tell whether the operation promised well or not. When cases could be selected statistics would probably be more encouraging and favorable.

The President thought that the theory of rheumatism in this case could not be entirely disposed of.

Dr. C. L. **DANA** said he did not share the optimistic views which Dr. Putnam had advanced, and should be sorry to start a boom in spinal surgery. If the history of all the operations already performed in this field was known, he thought they would all be disposed to follow the conservative suggestions of Dr. Sachs, and would only deal with selected cases. The operation was not, in the speaker's opinion, a simple one.

Dr. **GIBNEY** said that he for one did not by any means desire to start a boom in spinal surgery upon the results of a single cure. What he had meant by an easy operation was that in these old cases of Pott's disease the spinous processes were more prominent, and the soft parts were thin and readily got at. Cases should, of course, be most carefully selected; but when patients were evidently going down and the cases began to assume a serious aspect, he thought that surgical interference, properly undertaken, was justifiable.

Dr. DERCUM thought that the proper cases were difficult of selection, and it was only now and them that success could be hoped for. He regarded the result in his case as fortuitous.

Dr. G. M. HAMMOND presented a report on the "Pathological Findings in the Original Case of Athetosis," on which Dr. W. A. Hammond's description of athetosis was based. After briefly referring to the case, Dr. Hammond stated that the portion involved in the lesion consisted of fibrous connective tissue. Topographically, the lesion was a lengthy one in the antero-posterior direction, parallel in its short axis with the internal capsule. Its posterior end invaded the stratum zonale of the thalamus in its posterior third, and the posterior third of the posterior half of the internal capsule. In its anterior extension it crossed the capsule, invading the posterior third of the outer articulus of the lenticular nucleus. The author called attention to the fact that the motor tract was not implicated in the lesion, and claimed that this case was further evidence of his theory that athetosis was caused by irritation of the thalamus, the striatum, or the cortex, and not by a lesion of the motor tract.

Dr. E. C. SPITZKA reported a case of hemi-athetosis, in which the lesion was found to be in the same situation as the one in Dr. Hammond's case.

Dr. E. C. SEQUIN presented a paper on "Atheto-Choreic Spasm of the Right Side of the Body." The post-mortem showed a glioma of the left thalamus opticus and adjacent internal capsule.

Dr. Seguin's views were that all cases of athetoid and choreiform movements following hemiplegia were due to lesions involving the thalamus and adjacent capsule.

Dr. PUTNAM then read a paper on "A Form of Poly-Neuritis, Probably Analagous to or Identical with Beri-Beri Occurring in Sea-Faring Men in Northern Latitudes." (See page 495.)

Dr. GRAY asked if these ships had carried large quantities of ice, because he had seen cases of neuritis following the handling of ice, which had promptly subsided if this cause was removed. The condition might in Dr. Putnam's cases have so originated and then become infectious or contagious.

Dr. BIRDSALL inquired as to the amount of air supplied to the hold of these vessels, and to the seamen's quarters. Cases recently reported to him had led to the impression that the men who had died had been specially exposed to bad air in the hold, while those in good quarters had escaped. He agreed, also, that cold was an important element in the production of all forms of neuritis.

Dr. HERTER asked whether the men's diet had been of fish.

Dr. PUTNAM said that fish had not been the diet, but salt pork, sometimes fresh vegetables and fresh meat. But in one of the worst cases it had been salt pork.

Dr. BULLARD, of Boston, then read a paper on

DIFFUSE CORTICAL SCLEROSIS OF THE BRAIN IN CHILDREN.

Under this title he places those cases of diffuse cerebral sclerosis in which the cortical layers of the cerebrum are primarily or principally affected and where the microscope shows the presence in these layers of a homogeneous ground-substance or of a finely fibrous network with a few spider-cells in the meshes occupying the position of the neuroglia.

Accurate accounts of such cases with autopsies, in children, are rare. Doubtful whether cases of cortical sclerosis can always be distinguished from forms of diffuse sclerosis, in which the cortical layers are not specially affected. "From these we have not yet the evidence to determine in how far our cases differ."

Cases analogous reported:

Personal.—Boy, twelve years old; family history negative; always healthy; was bright, intelligent, did well at school. In August, 1886, had a fall, striking his head against curbstone and making wound over right parietal bone, of which the scar still remains. Wound suppurated, not painful, but some sensitiveness over head all winter. Three weeks after injury he forgot the baby, whom he had taken out-of-doors. (He was always fond of him, and was accustomed to take care of him.) Two months later he spent the whole night out, because he could not find his way home nor tell where he lived.

April, 1887.—Gradual loss of memory since accident; loss of interest in objects about him. Clonic spasm of tongue now ceased; clonic spasm of left face and arm. Has become awkward in use of extremities.

Present Condition.—Well developed and nourished; very forgetful and childish; markedly *weak-minded*. Sits much of the time with vacant expression; understands fairly what is said to him. Likes to sit by window and watch what goes by. Can read the name on a wagon, but attaches no meaning to the words. Speech slow, indistinct; occasionally omits words. Head of normal shape and size. Scar one and a half inch long over right parietal bone. Probable paresis of all extremities. Slight, but marked, inco-ordination, especially left. Sensation normal; knee-jerks exaggerated; waddles in walking. Double neuro-retinitis

Consultation with Dr. Russell.

Operation.—Trephining over cicatrice in right parietal region. Button of bone replaced. (Specimen shown.)

June, 1887 (ten months after the fall).—After operation, clonic spasms ceased temporarily, then gradually increased and involved all extremities. Aphasia; screams much. Is now almost helpless. Lies on mattress on floor; cannot get up alone, but can walk a little with assistance.

August, 1887 (twelve months).—Has had paralysis of sphincters for some time. Cannot speak; cannot sit up; cannot use hands or feet much. Morbid dementia; recognizes mother; inclined to pull things to pieces; grinds teeth.

November, 1887.—Left extremities almost totally paralyzed. Transferred to Boston Lunatic Hospital. Died in condition of extreme dementia (fifteen months after the accident).

Autopsy (made by Dr. Gannett).—Œdema of pia; chronic lepto-meningitis; atrophy of brain; chronic internal hydrocephalus, secondary to atrophy of brain; chronic ependymitis of fourth ventricle; no evidence of compression or obstruction of venæ Galeni. Brain did not quite fill cavity of cranium; weighed 1,260 grammes. Pia showed slight thickening and opacity; nowhere adherent.

Microscopic Examination. Pia showed a slight degree of thickening from growth of fibrous tissue. Brain: First layer of cortex showed the finely fibrous network with a few spider-cells in the mesh, due to atrophy of the nerve-fibres and an increase in the neuroglia. There was a slight degree of nerve-cell infiltration of the adventitial sheaths of the blood-vessels in the cortex. Beyond these nothing abnormal was observed.

The clinical picture presented (Moore's case and my own) is as follows: A varying time after a fall, intellectual impairment—at first simply obtuseness, general dullness, and loss of memory; then clonic spasms of face and extremities, which may later be followed by epileptic seizures. Loss of motor power, sometimes appearing suddenly as a hemiplegia, sometimes very gradually, but usually involving the extremities, on one side especially; sometimes inco-ordination of extremities with similar distribution. Speech affected, slow, thick, or aphasic, accompanying a right hemiplegia. Loss of power in extremities progressive, in no case absolute; involvement of sphincters. As the disease progresses, the spasmodic manifestations tend to increase in frequency and severity; epileptic attacks and opisthotonus may occur. Neuro-retinitis or optic neuritis. Sensory symptoms almost absent.

Progressive Failure of Intellect.—Simple rapidly progressive deterioration of all the mental powers, a pure dementia uncomplicated by intervals of excitement or violence. No hallucinations or delusions detected.

"The symptoms of this affection are those of a gradually progressive mental deterioration, uncomplicated by hallucinatory or delusional conditions and a gradually progressive loss of power, involving all the extremities, the trunk-muscles, and the sphincters, loss of sight, loss of speech all these accompanied by symptoms of motor explosion, clonic spasms, tonic spasms, and eventually epileptic seizures." Inco-ordination; absence of sensory symptoms. Neither tremor nor nystagmus in either of these cases.

Diagnosis.—The most important practical point is to determine whether we are dealing with a diffuse process or a local one. In certain cases this is by no means easy to determine. Where the explosive symptoms predominate and the paresis is slight or unilateral, and especially where, as in my case, a cicatrix exists over the motor region on the side opposite to that of the hemiparesis, the possibility of a localized starting-point cannot be excluded, although the marked mental enfeeblement, in conjunction with the motor symptoms, would point to an affection of considerable extent, and would suggest the existence of a subacute or chronic form of lepto-meningitis. It is the possibility of a localized lesion or new growth, accompanied by a chronic or subacute inflammation of the meningeal membranes, which may, at least for a time, render an absolute diagnosis impossible. "A cerebral tumor, uncomplicated by meningitis, would be very unlikely to produce this combination of symptoms."

Differential diagnosis from multiple sclerosis; from congenital and early forms of cerebral atrophy; from all forms of lobar or partial atrophy; from primary hydrocephalus.

Provisional Conclusions.—There exists in children a non-congenital form of diffuse cerebral sclerosis in which the cortical layers of the brain are more specially affected, and which is distinguished from the other forms by its appearance in healthy children, either without known cause or after traumata, by the steadily progressive character of its symptoms and by the especial prominence of the gradually increasing *dementia*, which finally reaches an extreme degree without a corresponding loss of motor power, and while the sensation is comparatively unaffected.

[TO BE CONTINUED.]

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, May 6, 1890.

THE President, DR. LANDON CARTER GRAY, in the chair.

Dr. LANDON CARTER GRAY read a paper, entiled: "Can We Diagnose Hyperæmia and Anæmia of the Brain and Cord?" After citing the views ot such recent writers as Gowers, Strümpell, Liebermeister, Ross, Seeligmüller and Hammond, and showing that they all, with the exception of the one last named, spoke with considerable skepticism of the possibility of diagnosis, the writer then passed in review the symptoms upon which the diagnosis is supposed to be made. Of these cephalgia, flushing and pallor of the face, vertigo, tinnitus aurium, insomnia, delirium, were, he said, general symptoms which can only derive diagnostic value from their associated symptoms. Slight retinal changes of vascularity are almost impossible to differentiate from the normal, as every well-informed opthalmologist knows, and more marked retinal changes, such as neuro-retinitis, are indicative of organic intracranial disease. Myosis and motor and sensory paralysis have never yet been shown to have been caused by simple hyperæmia or anæmia of the brain. If the symptoms of apoplexy, paralysis, convulsions, a soporific condition, mania and aphasia, which Dr. Hammond states to be indicative of cerebral hyperæmia and anæmia are symptoms of organic intrecranial disease, and the pathological data which are given by Dr. Hammond, such as enlarged and dilated blood-vessels, increase in consistence and density of the white matter, red or violet hue of the gray matter, subarachnoid or ventricular or choroid effusion, hematine, the *l'état criblé*